Prcewosky is the only pathologist who found diverticula (not due to traction) in the lower part of the œsophagus. These diverticula, however, were not larger than a hazelnut. We cannot be too particular in making an accurate diagnosis of such rare conditions.

Reitzenstein, after investigating the cases of Strümpell and Klemperer, rejects them as not being true diverticula, and takes into consideration only the cases of Mintz, Reichmann, Kelling and his own. Mintz's case demonstrates distinctly the two different "swallowing sounds." His patient vomited matter of the previous day without bringing up the food eaten the same day. Mintz did not make Rumpel's test with the two tubes. Neither did Reichmann make use of his test. His first case was examined only superficially, and must, therefore, be considered as doubtful; his second case furnishes better proof. An œsophageal sound showed the passage into the stomach to be free after the diverticulum had been emptied of its contents. When pouring in water to the amount of 400 c.c. about 300 c.c. ran through into the stomach, while 100 c.c. remained in the diverticulum.

Kelling mentions that Mercier's œsophagus sound has been used and appreciated only by Leube. Kelling's case has been thoroughly examined and described. He arrived at his conclusions in a way different from Rumpel by making use of the second "swallowing sound." His résumé is as follows:

1. If the tube slips easily into the stomach, one can exclude ring-shaped, anatomical strictures.

2. If the tube does not penetrate at all into the stomach, the contents of the œsophagus are removed and a stiff tube is introduced. This is closed at the end and has one perforation at the side. Through this tube lukewarm water is poured in, and if by auscultation gurgling sounds at the cardia can be recognized, the attempt is made to push the tube further down. If, while the gurgling sound is audible at the cardia, the tube slips readily into the stomach the case is one of spasm. If, however, in spite of the running in of water the tube does not go further down the probability is that there is no spasm.

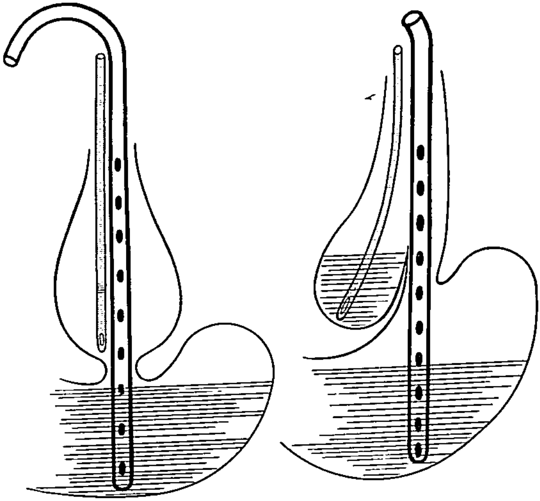
3. If the tube is arrested in the œsophagus and remains immovable in this position for a while, and then suddenly slips into the stomach, it proves the presence of spasm.

The presence of the swallowing sound is not an infallible test, however, for there are cases:

(*a*) Where the position of the œsophagus sound or its change from one position to another cannot be demonstrated.

(*b*) The second "swallowing sound" may be absent in diverticula, as Reitzenstein and Reichmann's second case have shown. On this point Kelling's method of diagnosis cannot be relied upon. The second swallowing sound is not usually present in cases of dilatation with spasm of the cardia. In the forty-one cases reported the sound was either missing entirely or heard much later, or else was of a different character. It may also happen that the second sound immediately follows the first, as in Westphalen's case. Sometimes the two swallowing sounds can be heard at the back and not at all at the sternum. Again, only the first sound may be heard at the sternum, and not the second. Westphalen describes the sound which immediately follows the first gurgling sound as though a fluid were pouring into a pot partly filled with water. This was also the character of the sound recognized in my first case at the back, but not at the sternum.

FIG. 1.



Rumpel has recently made use of a most valuable test to show the difference between diverticulum and dilatation. He employs two tubes, one with numerous perforations in its lower third is passed directly into the stomach, the other into the œsophagus above the cardia (Fig. 1). Water, which is poured through the œsophagus tube, will run down into the stomach through the perforated tube in case of a dilatation. In the event of a diverticulum this will first be filled, and only the overflow will run into the stomach. The contents of the diverticulum can be

1. If one introduces the double tube into the stomach one can readily aspirate gastric juice through the inner tube. (Should this tube be very thin the gastric contents can be diluted by pouring in a definite small quantity of water.)

2. The stomach can be filled and emptied at will.

This water test can now be made without in the least interfering with the rest of the experiment. With Rumpel's perforated tube a return flow of water cannot take place. If water is now poured through the œsophagus tube:

FIG. 2.

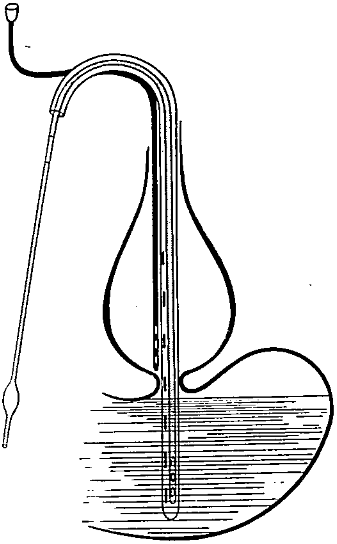
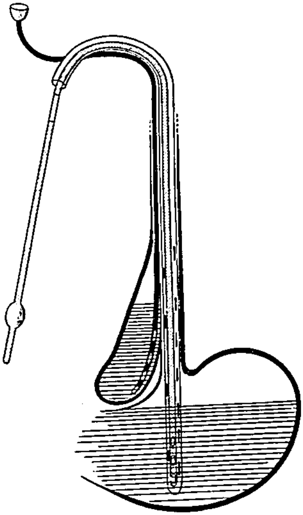


FIG. 3.



a. In case of dilatation the water will run through the perforation of tube No. 1 into the stomach, the inner tube being no obstacle. Besides this, the inner tube can be removed after having accomplished its purpose of aspirating gastric juice (Fig. 2).

b. In case of a diverticulum a certain amount of water will remain in the pouch, and can be returned through the œsophagus tube (Fig. 3).

The inner tube may be made of soft metal, thereby giving a certain amount of stiffness to the outer tube. One could also give it the bend of Mercier's sound. This latter would be necessary to satisfy Kelling's demands. My own experiments were made by means of a rather stiff stomach tube and an inner tube 65 cm. long, on the order of a ureteral catheter. This inner tube has sufficient firmness to be pushed in and out as necessity arises. The patient swallows the perforated stomach tube, and I afterward introduce the inner tube. If the entrance to the stomach is difficult to find, the double tube, with the inner tube of soft metal, to which is given the curve of Mercier's sound, is introduced. If by this method of experimentation it has been proved that the perforated tube is in the stomach, no water ought to return from it, at least not before the stomach is filled to overflowing.

Reitzenstein does not lay sufficient stress upon this fact.

I found that every time when water could be returned through the perforated tube this latter was not in the stomach, but bent upon itself in the œsophagus.

CASE I.—On June 22, 1897, a man came to my office in a state of exhaustion. It was believed that he had a cancer of the stomach. He had lost twenty pounds during the last few months. He could not eat, and could swallow only with the greatest difficulty. Food could not be retained. Besides, he was subject to the most painful cramps in the pit of the stomach, for which physicians had resorted to morphine. He was determined not to continue this drug on account of its unpleasant effects. As the patient complained of difficulty of swallowing for four years, and had been subject to cramps previous to that time, the diagnosis of carcinoma was questioned.

W. W. P., captain of police, is sixty-four years of age. His father died of old age at eighty-four years. Mother died of typhoid fever. No hereditary taint. Patient had in his youth suffered from typhoid, variola, spotted fever, and malaria. He developed, however, into a strong, robust man. He admits that he eats hastily, and his daughter relates that he gulps down whole tumblerfuls of ice water at a time. In his younger days he used to smoke from fifteen to twenty cigars a day. When about forty years of age he commenced to suffer from fulness after eating. Soon after this cramps developed, which reached an intense degree. Patient describes these in the following manner: When least expected he suddenly experiences a slight pressure in the pit of the stomach. He knows at once what this signifies, drives home as quickly as possible, and goes to bed. The pain intensifies so rapidly that the patient has to press hard with both hands on his stomach in order to get some relief. At the height of the attack he is not able to breathe, and almost chokes. By degrees the contraction subsides, and he is able to breathe again with peculiar sighing sounds. The pain seems to cut off the respiration, as he expresses it. Physicians have given him injections of morphine, and the patient chloroforms himself. Every imaginable diagnosis had been made: spasm of the finest bronchioles, asthma, liver colic, gallstones. During all these years the patient had dyspepsia, but did not vomit. About six years ago he commenced to raise food, and at the same time the fulness after eating diminished. At first the vomiting seemed to ease his pain, but later it remained the same. The vomited matter was often mixed with mucus. Patient was not able to tell whether the ejected food came from the œsophagus or from the stomach. He always called it "vomiting." After two more years of this condition the patient became almost unable to swallow. He could help himself, however, by taking water between each mouthful. Later this always failed. Patient often ejected his whole meal over the table in the midst of such a swallow. He commenced to eat alone, and became melancholic and despaired of medical aid, as the "physicians prescribed remedies for him, but no one could teach him how to get them into the stomach." Patient lost flesh rapidly. Only during a short time he got a little relief while a physician had attempted to pump him out; but this was soon abandoned. Shortly before the patient came to me he had been through one of his fearful attacks of cramps. When he had diarrhœa he always knew that an attack was coming on. Whenever he felt it approaching he had an irresistible desire to lie down. Again, he was much troubled at night by coughing, and had to vomit mucus before he could find rest. The only position in which he could sleep was with his thorax raised. Sometimes he is better able to swallow by putting his head as far back as possible. He feels that the food meets with resistance before passing into the stomach. At such times, then, there cannot be spasm of the cardia, but another obstacle higher up (as found later this obstacle was the entrance of the diverticulum).

Status. Height, five feet eleven and one-half inches; man with large frame, faded skin, heart sounds normal, pulsus tardus, but alike at both wrists; no pronounced arterio-sclerosis; patellar reflexes not increased; no dulness along the spinal column.

Upon introduction of the stomach tube about 300 c.c. of stagnating food came out. At a distance of 41 cm. from the teeth the tube met with a resistance. After repeated attempts with large tubes the stomach was finally reached. This was proved by the analysis of the matter brought up. The fact that the largest sounds passed the obstacle easier than the thin ones, together with the long duration of the disease, decidedly excluded carcinoma.

The stomach splashed as low as two fingers below the navel. Liver was not enlarged. Urine contained neither albumin nor sugar. The material withdrawn from the œsophagus was usually of a sweetish odor, in three layers, and mixed with much mucus. All subsequent examinations showed the same characteristics and large quantities of food of the previous day. Spinach was especially easily recognized, and had sometimes been eaten two days before. I succeeded in putting a Boas test-meal into the stomach through the tube. After an hour it was removed, and showed the following analysis:

Total acidity, 30; free HCl, 0.036 per cent.; congo, phloroglucin vanillin. Tropæolin gave a positive result; pepton and propepton present; rennet ferment present; rennet zymogen; milk coagulates until a dilution of 1:80 is reached. Sugar test weakly positive; starch and achroodextrin present; lactic acid present in small quantity.

The analysis of the contents of the œsophagus on the same day showed the following:

Total acidity, 11; congo test, weakly positive; phloroglucin vanillin; tropæolin reaction absent; no biuret reaction; no rennet ferment; no rennet zymogen; sugar reaction strongly positive; lactic acid strongly positive.

The opening into the stomach frequently could not be found. Besides, the patient could not bear much manipulating because of great pain. When I tried to pass thick elastic tubes, in order to let them remain for some time, I had to give it up on account of supervening cramps. At other times the patient suddenly felt the tube slip as if into the stomach. It conveyed the same sensation to my fingers. Usually, however, I could not pour water through the tube, or, if so, very little. Matter brought up showed no HCl or ferments. The tube simply had bent into a curve and remained in the space above the stomach. If the tube really was in the stomach it became an easy matter to pour water in and out. If a weak solution of eosin was put into the stomach and a solution of methyl-blue through a second tube into the œsophagus, both solutions could be returned with their original color. The amount of food in the œsophagus in the morning varied between 210 to 300 c.c. The microscopical examination of the œsophagus contents showed:

Vegetable cells, fat globules, starch granules, yeast in large quantity, bacilli; a very active variety of coccus.

In order to make an accurate diagnosis between dilatation and diverticulum or dilatation with diverticulum, I made my test with the inner and the œsophageal tubes, as described above. After the œsophagus was emptied of its contents I tested the presence of tube No. 1 in the stomach. The inner tube was very readily passed in and out for this purpose. The contents of the stomach, as brought up through this inner tube, showed the following analysis (three different experiments): Total acidity, 80, 100, 150; free HCl, 0.18, 0.14, 0.328; congo, phloroglucin vanillin, tropæolin strongly positive; ferments positive.

The contents of the œsophagus during the same experiments showed the following reaction: Total acidity, 26, 21, 20; free HCl, none; no ferments; sugar test positive; lactic acid reaction strongly positive.

Whenever 400 to 500 c.c. of water had been poured in about 180 to 225 c.c. could be returned through the œsophageal tube introduced to a distance of 45 cm. The rest of the water could be returned through the inner tube from the stomach. The degree of acidity of these two fluids was always very different. Both were acid, but the œsophagus water reacted feebly upon congo, while the stomach water showed a marked reaction. If the œsophagus was filled to the limit it could hold 500 c.c. About 200 c.c. of this, as shown above, were contained in the diverticulum. The gastrodiaphane showed an illuminated zone at the processus ensiformis sterni, which did not correspond to the level of the stomach (Fig. 4, A). This transparent zone was situated above the upper border of the stomach, which could be distinctly demonstrated by inflation. The electric light could be pushed below this point and to the left at other times, transilluminating the stomach proper (Fig. 4, B). The examination by means of the X-rays did not give very satisfactory results as far as details are concerned. Yet a very useful point was determined. The accompanying sketch (Fig. 5) gives a posterior view, the broadening of the shadow corresponding to the spinal column is caused by the dilatation of the œsophagus. The distinct bulging to the right shows the location of the diverticulum. I had filled the œsophagus with a bismuth solution and also made the

FIG. 4.

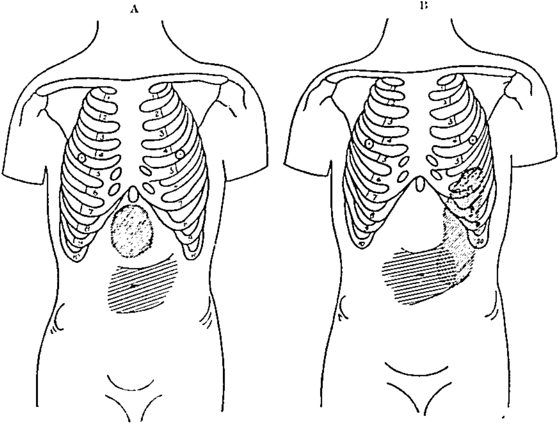
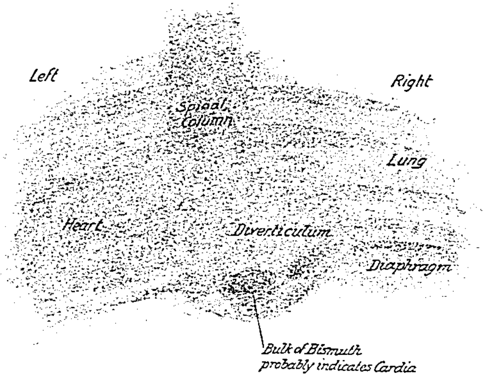


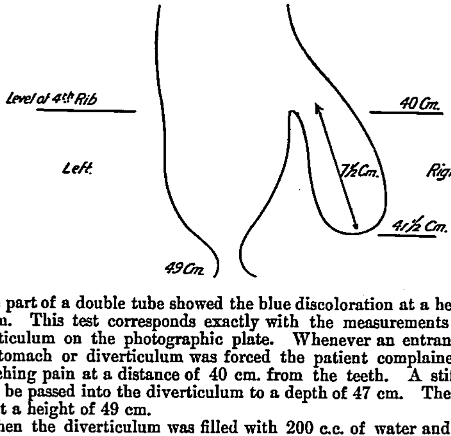
FIG. 5.



patient swallow some bismuth tablets. Besides this I had introduced a lead sound. The latter is scarcely visible in the picture. The bismuth tablets show most distinctly as the darkest spot on the positive. It was interesting to notice the motion of the diaphragm before we exposed fifteen minutes. These excursions extended over 5 cm. By subtracting these from the height of the diverticulum, thus correcting the enlargement of the shadow caused by the respiration, we obtained 7½ cm. as the actual height of the diverticulum. The skiagram shows distinctly that the diverticulum was situated above the diaphragm.

The oscillations in the manometer test were inverse in the stomach to those in the œsophagus. This also proved the diverticulum to be above the diaphragm. The opening of the diverticulum could not be found by means of the test used by Kelling. I could hear only the first swallowing sound at the sternum. The second sound never could be heard in all my experiments. Immediately following the first sound at the sternum there could be heard a second at the back, such as Westphalen has described.[1] It sounded as though liquid fell into a pot which already contained liquid. This sound was not always audible. If the diverticulum was filled with 200 c.c. of a solution of methyl-blue the outside white part of a double tube showed the blue discoloration at a height of 7½ cm. This test corresponds exactly with the measurements of the diverticulum on the photographic plate. Whenever an entrance into the stomach or diverticulum was forced the patient complained of a scratching pain at a distance of 40 cm. from the teeth. A stiff tube could be passed into the diverticulum to a depth of 47 cm. The cardia was at a height of 49 cm.

FIG. 6.



When the diverticulum was filled with 200 c.c. of water and a connection made with the outside in the manner of communicating tubes, the entrance of the diverticulum was indicated by the level of the fluid at the height of the fourth rib. The above picture was obtained (Fig 6).

The subjective feelings of the patient corresponded exactly to these measurements. At X the first resistance was met, and only at X the two separate openings had to be forced. This obstacle once passed, a soft tube could bend itself as well in the œsophagus above the cardia bring up large quantities of slimy water. At night this fluid often came even through the nose if her head was low. Twelve years ago she began to experience difficulty in swallowing. The attending physicians were unable to recognize the nature of the trouble, and her family despaired of her recovery. She lived on rice and cream, but the attacks did not diminish. Her symptoms had come one by one in the course of years. First painful stomach cramps, then vomiting without nausea, and, lastly, difficulty of swallowing. Patient is able to state very accurately whether food reaches the stomach or not. The pressure in the latter case is very painful, especially over the left side under the breast. The heart beats with a violence almost unbearable, and her arms and hands are cold as ice. Mrs. S. is obliged to walk up and down with her arms raised above her head and holding her breath. Suddenly the inside resistance seems to yield, she feels the food rush down, and the attack is over. Patient can now return to the table and eat, although usually too exhausted to do so. Cold beverages are her worst enemies. A tumblerful of ice water at meal-time invariably brings on an attack, which Mrs. S., like the first patient, compares to choking. Between meals the patient is able to drink, yet has to be very careful. Mrs. S. is absolutely sure that often the ejected food is a day old, sometimes even two days. She also noticed that it is not at all mixed with the last eaten food. She very rarely vomits from the stomach. At night she is troubled with coughing and prevented from sleeping. She cannot lie down flat. Carbonic acid gas in any form gives her the greatest relief. Sometimes the drinking of large quantities of water produce vomiting, and thereby relief. Patient is of a nervous temperament. She considers the severe malarial fever of her youth the cause of her trouble. Of course, she did not suspect the real nature of her disease.

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[1] Westphalen: Archiv für Verdauungs Krankheiten, Band v., Heft 1.

Status. Small size; weight, 130 pounds. Mild anæmia. Patellar reflexes somewhat exaggerated, no hyperæsthesia, delicate constitution. Heart normal, pulse 72, goes to 85 as soon as patient is excited. It is an easy matter to raise up 100 c.c. slimy food remnants from the œsophagus. At the level of the cardia one meets with an obstruction which cannot always be overcome. At times one succeeds in getting into the stomach, as shown by subsequent analysis. The contents of the œsophagus never showed free HCl or ferments, while lactic acid and sugar were always plentiful. With much difficulty I succeeded in getting a test-meal into the stomach proper. The analysis after an hour was as follows: Total acidity, 40; free HCl, 0.07 per cent.; starch, achroodextrin; lactic acid, weak; ferments, positive.

A diagnosis of cardio-spasm with dilatation of the œsophagus was made. As soon as the tube reached the cardia a most painful spasm occurred. Elastic solid sounds could not be used at all. Sometimes the use of the tube had to be abandoned, because a cramp would occur as soon as the lower end of the œsophagus was reached. Quite accidentally we found that it was not always necessary to resort to morphine or chloroform to overcome the painful spasm, as a small Seidlitz powder would do the same, and the effervescing bromo-water proved especially effectual. The carbonic acid gas seems to have a decided effect upon the innervation of the muscle, as is also the case in atony of the stomach. Very small quantities were sufficient to secure prompt relief. In severe cases I give first a solution of sodium bicarbonate, and follow it by one of acid tartar. My first case was thereby entirely relieved from the have shown. In the differential diagnosis of diverticula it will be well to consider the following points:

1. With dilatation as well as with diverticula there occur painful cardio-spasms (Kelly, Jung) as well as painless cases (Reitzenstein).

2. In both vomiting occurs from the œsophagus, occasionally from the stomach. Even in cases of typical cardio-spasm eructation from the stomach may occur.

3. If food is vomited, eaten one or two days before, while other food eaten in the meantime remains down, the case seems to be one of diverticulum. No observations to the contrary have been made so far.

4. The swallowing sounds are abnormal in all cases of dilatation. In cases of diverticula the swallowing sounds are audible in 33 per cent., and can be used for diagnostic purposes (Kelling's method). Westphalen's sound can also be heard in diverticulum with dilatation of the œsophagus (Jung).

5. If after emptying the œsophagus it is easy to enter the stomach a diverticulum probably exists (Mintz, Reichmann). Yet the passage of the tube may be difficult. "Only once out of ten experiments does one find the cardia and gain entrance into the stomach" (Reitzenstein).

6. In making Rumpell's test, proof must be furnished that the tube is actually in the stomach. Inflation in this case is impossible, as feeling of the sound is not always practicable.

7. The proof positive of having entered the stomach is obtaining gastric juice through the inner tube (Jung).

8. Mercier's sound or Leube's diverticulum tube are necessary in order to find the entrance into the stomach. These tubes must be perforated for the diverticulum test, and by means of a thin inner tube gastric juice can be brought up.

9. After a diverticulum has been diagnosed its height can be ascertained (*a*) by Kelling's test (swallowing sounds) and (*b*) by filling the diverticulum with a colored solution and putting a narrow strip of white adhesive plaster longitudinally on the tube.

10. Neubauer's experiment for ascertaining the level of the liquid can also be used according to Kelling to determine the entrance into the diverticulum.

11. The entrance of the diverticulum can easily be overlooked by œsophagoscopy.

12. Gastrodiaphany did not show the diverticulum in Mintz's case, and gave no information about the kind of enlargement. In Reitzenstein's case it was possible to make the diagnosis diverticulum by gastrodiaphany alone.

13. X-rays show an enlargement of the œsophagus only.

14. The statements of even the most experienced patient, that the tube is in the stomach, are often erroneous. The bending of the tube

gives the patient and physician alike a sensation as though the tube had entered the stomach.

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THE CLINICAL ASPECT OF PLAGUE.

WITH ILLUSTRATIVE CASES.

BY FRANK G. CLEMOW, M.D. Edin., D.P.H. Camb.

I.

IT can scarcely be claimed that the last word has yet been said upon the terribly fatal disease which has caused such ravages in India and elsewhere during the past few years. Little apology is therefore needed for any further contribution which may tend to throw light upon the nature of the malady, and I gladly comply with the request of one of the editors of this JOURNAL to communicate an article upon the subject.

The following notes are an attempt to put upon record the clinical facts of plague as they have been observed by myself in a large number of cases. They do not lay claim to be in any sense a full and complete description of the disease. Considerations of space alone would have prevented the undertaking of so great a task. They are confined entirely to the clinical aspect of plague. The etiology, bacteriology, mode of spread, pathology, prognosis and treatment of the disease, if alluded to at all, are mentioned only incidentally and as briefly as possible, and where it seemed necessary to refer to them in illustration of the clinical appearances.

The general character of plague and the principal symptoms and signs which usually characterize an attack of the disease have been so repeatedly described that it appears unnecessary to give any preliminary account of them before commencing a discussion in some detail of the clinical phenomena of the disease.[1]

Four principal varieties of plague have been recognized, depending upon the channel by which the bacillus associated with the disease enters the tissues of the patient, and the clinical features in each instance are to a great extent modified by this primary factor in the production of the disease. The four varieties are the following:

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[1] The present paper will be based principally upon the clinical notes of 502 cases of plague admitted to the Parel Old Government House Hospital, Bombay, during the nearly three months the author was in charge of it, in addition to notes of 152 cases already in the wards at the commencement of that period, and of a large number of cases seen in the hospitals and in district work in Calcutta.

1. *The bubonic variety of plague,* in which the bacillus appears to enter the body through some abrasion or discontinuity in the skin, and passes at once to the nearest lymphatic gland or glands, where it proceeds to multiply, causing in consequence inflammatory swelling of the gland or glands involved.

2. *The pneumonic variety of plague,* in which the bacillus gains primary access to the lungs and produces a specific affection of these organs, of the nature of a lobular pneumonia.

3. *The septicæmic variety of plague,* in which the bacillus probably enters the system in the same manner as in the bubonic variety, but, instead of remaining for a time in a lymphatic gland and producing a bubo, appears to pass almost at once into the general lymph stream and to affect a very large number of glands throughout the entire body. As the word septicæmia implies, the bacillus in these cases probably gains access to the blood-stream very rapidly—more rapidly than in the bubonic variety of the disease. Fatal cases of the latter and of the pneumonic variety very frequently, if not always, become septicæmic before death; that is to say, the bacilli escape from the gland in one case and the lung in the other and enter the general blood-stream. The term septicæmic, as applied to the cases included in the group under discussion, is, perhaps, a not altogether satisfactory one, for there is good reason to believe that the bacillus never grows primarily in the blood-stream; it appears to require a preliminary stage of growth, either in the lungs or in some part of the lymphatic system—whether in one or many glands—where it can produce a sufficient quantity of toxins to overcome the protective forces possessed by the blood against an invasion of bacilli. This is a pathological question which it would be out of place to discuss more fully in the present paper, where it is intended to deal only with the clinical manifestations of the disease.

4. *The abdominal, or enteric, variety of plague.* A group of cases have been described in which the symptoms of gastro-intestinal affection are unusually marked, and in which it has been thought that the bacillus has entered the body by means of the gastro-intestinal tract, either in food or in drinking-water. It will be shown, in discussing the phenomena of plague in connection with the alimentary system, that abdominal symptoms are in some cases unusually prominent, but the proof that these cases are produced in the way described is wanting, and, for the present, it must remain an open question whether this mode of infection does occur in the human body.

Of these four varieties of plague the bubonic is, in most epidemic outbreaks, by far the commonest, cases of this kind contributing from 80 to 90 per cent. of the total cases. It is to this variety of the disease that the following notes in the main refer, incidental reference only being made to the other varieties mentioned. In these notes, I should wish to add, only the clinical features proper to an attack of plague are dealt with; considerations of space alone prevented a discussion of the innumerable complications and sequelæ which are so commonly associated with the disease.

THE TEMPERATURE. The temperature-chart in a case of plague shows no such characteristic curve as is commonly met with in many other febrile diseases, such, for example, as relapsing fever or ague. The rise from the normal is probably more or less rapid, but in hospital practice in India it is rare to see a plague patient during the period of onset, and most usually at the time of admission to hospital the fever has already attained a considerable height. More often than not the temperature is over 103° on admission, and temperatures of 104°, 105° and even higher are not very uncommon. In perhaps the majority of cases the temperature rises at some period in the course of the attack to over 104°. In some cases it may rise before death to 106°, 107° or 108°. These high temperatures are usually met with immediately before death; but this is not invariably the case, and death may be preceded by a marked fall of temperature even down to or below normal.

The following figures may help to throw some light upon the relation of the height of the temperature to the severity of the attack. They show the percentage mortality in cases grouped according to the highest point attained by the temperature at any period of the illness. In the first two groups, it should be noted, the thermometer rose to 104° or 105° on more than one occasion in some of the patients. In the last three groups the temperatures of 106°, 107° and 108°, respectively, were reached on one occasion only in every case.

TABLE I.

Highest temperature at any period of attack.	Number of cases.	Died.	Percentage mortality.
Between 104° and 105°	217	187	86.18
" 105 " 106	68	57	83.8
" 106 " 107	13	12	92
" 107 " 108	1	1	100
108°	1	1	100
Total above 104°	300	258	86

The percentage mortality among the 300 patients in whom the temperature rose above 104° was 86. On the other hand, in 202 patients under treatment at the same time and under exactly the same circumstances, but in whom the temperature did not at any time rise as high as 104°, the mortality was only 73.7 per cent. It would seem, therefore, that a rise of temperature to above 104° F. is a distinctly unfavorable sign. It is somewhat remarkable that the mortality in the second group (patients whose temperature rose to between 105° and 106°) was somewhat lower than that in the first group (patients whose highest temperature was between 104° and 105°). The safest conclusion to draw from these figures—and it is one supported by general impressions—would appear to be that while a temperature of 104° or over is an unfavorable prognostic sign, a rise to 105° is not more unfavorable than a rise to 104°, but that above this point each further rise of temperature implies a more severe attack of the disease and a graver prognosis.

Of 15 cases in which the temperature rose above 106°, only 1, it will be observed recovered, while of the two patients in whom the temperature rose above 107° and 108° respectively both died. The following are brief notes of three of the cases just referred to:

CASE I. (Register No. 757. Recovery after a rise of temperature to 105°.)—Male, Hindoo, aged eighteen years. Left axillary bubo. Vomiting and delirium troublesome. Secondary pneumonia, and rise of temperature to 106°. Admitted on fourth day of the disease, and discharged, cured, thirty-five days later.

CASE II. (Register No. 981. Death after rise of temperature to 107.8°.)—Hindoo, female, aged fifteen years. Right axillary bubo. Much delirium. Some bronchitis. Admitted the seventh day of the disease, and died eighteen hours after admission. Temperature just before death, 107.8°.

CASE III. (Register No. 951. Death after rise of temperature to 108°.)—Hindoo, female, aged eighteen years. Left axillary bubo. Much vomiting. Excessive delirium. Albumin in urine. Bronchopneumonia and rise of temperature just before death to 108°. Admitted on the sixth day of disease and died twenty-eight hours after admission.

Perhaps the sole characteristic common to the majority of temperature-charts of plague is a marked irregularity in the curve. There is very commonly a tendency to a morning fall and an evening rise in temperature, but rises and falls in temperature may occur at any period of the day. Sometimes it is possible to trace the rise to some definite cause, such as the onset of secondary pneumonia or the occurrence of suppuration in the bubo, but quite as commonly a rise in temperature may occur without any obvious cause. Sudden falls of temperature, equally without apparent reason, are by no means uncommon, and may occur at any hour of the day. A fall of some degrees on the second or third day of the attack is not infrequent, and has been regarded by some as a feature characteristic of the disease; it is, however, very often absent. In some cases falls of temperature can be definitely traced to a cause, such as the occurrence of collapse from heart failure, but in a large number of cases no obvious cause can be discovered. A knowledge of this fact is of importance in judging of the efficacy of certain forms of treatment, as it is quite possible that falls of temperature ascribed to the particular form of treatment would have occurred under any other treatment or with no treatment at all.

At the same time there is no question that high temperatures in plague can, with very considerable ease, be artificially reduced by appropriate remedies, such as antipyretics, the hot- or cold-pack, and diaphoretics. In view of the facts above stated as to the relation of the height of the temperature to the severity of the disease, it seems a rational plan to endeavor to reduce the temperature when it rises to a dangerous height. The method may not, perhaps, be based upon quite faultless reasoning, for it may, with full justice, be argued that the high temperature which is so frequently followed by death is not in itself the danger or the cause of death, but merely an index of the tissue and other changes resulting from the action of the toxins or bacilli, or both, circulating in the blood, and that merely to reduce the temperature is not to strike at the cause of the disease. But, on the other hand, it will be found in practice that an artificial reduction of temperature does not only relieve the patient from much suffering, but also appears to in some degree influence the course of the disease for good. The method I have most usually employed is the administration to any patient whose temperature rises above 104° of fifteen grains of phenacetin dissolved in hot rum and water; immediately after the dose the patient is placed in the hot-pack, and perspiration may be further encouraged by a dose of spirit of nitrous ether. Any depressing effect which the large dose of phenacetin might have had appeared to be counteracted by the hot spirit in which it was given. The hot-pack was invariably used in preference to the cold, and I have no personal experience of the effects of the latter. The indications of the disease would certainly point rather to the use of the hot- than of the cold-pack. The skin is usually excessively hot and dry, the functions of the sweat-glands are inhibited, the internal organs are frequently congested, and the kidneys more or less affected, if not in a condition of nephritis. Under these conditions, with an overwhelming quantity of toxins or bacilli, or both, circulating in the blood, it would appear most desirable to encourage in every way the action of the sweat-glands, and the application of an ice-pack would seem, *à priori*, to be fraught with no little danger. No doubt a more rapid reduction of temperature can be effected by the latter method, and it is right to add that in some other hospitals, notably in the Arthur Road Hospital, Bombay, the cold-pack was almost invariably employed, and, it was stated, without any unfavorable results. Native plague patients, however, greatly dislike this form of treatment, and unless very clear benefit can be proved to have been derived from it, this fact may well be taken into consideration, and the patient spared such unnecessary discomfort as the application of an ice-pack implies.

Fears have been expressed as to the wisdom of reducing a high temperature in plague by artificial means, as in the course of the disease the sudden falls of temperature which occur independently of treatment are by some regarded as of unfavorable prognostic import. I am far from feeling sure that such falls of temperature are rightly so regarded. In a disease such as plague, where the mortality ranges between 70 and 90 per cent., and death is therefore the rule rather than the exception, it is easy to regard almost any indifferent but fairly common feature of the disease as of unfavorable import, and this has, I think, been the case in regard to falls of temperature. Such falls certainly occur in cases which recover, and unless the fall is very great, and the result of some obviously unfavorable cause, such as collapse from sudden heart failure, there would seem to be no reason to regard falls of temperature as of necessity bad prognostic signs, or to refrain from producing them by artificial means.

Occasionally a very extreme fall of temperature, accompanied by an excessive degree of collapse and heart failure, is recovered from. The following is the most remarkable instance of the kind that has come under my own observation.

Case IV. (Register No. 917).—Hindoo, male, aged twenty-seven years. Admitted to the Parel Hospital, Bombay, March 19, 1898, with a history of two days' illness. The case was a fairly typical one of plague, with right axillary bubo. There was troublesome vomiting. The urine was free from albumin. On March 21st the temperature at 6 P. M. was 103°. At 8 P. M. I was hurriedly summoned to the ward by an attendant, who reported that the patient had thrown himself out of the window. This was easily proved to have been impossible, and what had obviously happened was that the patient had got out of bed, had descended a steep staircase which was quite near the ward, and, after walking some little distance along a veranda on the ground floor, had collapsed from heart failure. Almost immediately after he was missed, and on search being made he was found lying where he had fallen, on the veranda. He was removed to bed, where I found him deeply collapsed, quite unconscious, pulseless, and to all appearances moribund. The temperature had then fallen to 98.4°, and at 6 o'clock the next morning it was only 95.8° (or 7.2° lower than it had been on the previous evening). The treatment was confined to the free exhibition of stimulants and the application of warmth, and, contrary to all anticipations, he began rapidly to improve from the time of the *contretemps* described. There were five loose motions during the night. At 9 o'clock next morning he was conscious and answered questions rationally, but had no recollection of the previous night's incident. The tongue was cleaner, but very pale; the pulse was extremely weak, the speech slow and thick, the vomiting had ceased and did not recur; the urine was still free from albumin; there was some cough, but the chest was normal. On the following day (March 23d) the general condition was greatly improved and the pulse much stronger; the temperature varied between 96.4° and 98°. On the 24th the bubo in the right axilla had completely disappeared. Convalescence was uninterrupted, and the patient was discharged, cured, on April 12th.

In cases which recover, the temperature falls to the normal gradually in the course of two, three or more days, rather than suddenly; in other words, resolution of the disease is usually by lysis and not by crisis. Occasionally, however, recovery is preceded by what appears to be a critical fall of temperature. Convalescence may be interrupted by more than one rise of temperature, due either to some obvious cause, such as the appearance of a fresh bubo, or the onset of some intercurrent affection, or due to some cause which baffles all efforts to discover. In malarial subjects suffering from plague repeated rises of temperature may occur throughout the illness and well on into convalescence, and in such cases the temperature-curve may be of a somewhat puzzling character unless the possibility of malaria be remembered.

THE ALIMENTARY SYSTEM. The state of the *tongue* in plague varies very considerably. Perhaps the commonest and most characteristic condition met with is one in which the dorsum of the tongue is loaded with a white or grayish-white fur. This coating of fur is usually dry on the surface; sometimes it is remarkably smooth, and when the smooth fur is, as it occasionally is, moist on the surface, the tongue may present an appearance somewhat remotely resembling mother-of-pearl—an appearance which some observers have described as characteristic of the disease. However thick the fur may be on the dorsum, it is very noteworthy that the tip and edges are, in the great majority of cases, clean, pink, and free from fur. The edges of the tongue are very frequently indented by the teeth.

While the characters just described may be regarded, for convenience sake, as those most typical of the tongue in plague, it is of great importance to remember that in a very large minority of cases the tongue may be absolutely atypical. In not a few cases the tongue is remarkably little affected and only covered with a very thin coating of white or yellowish fur, through which the papillæ may be very clearly visible and apparently unusually prominent. In a rather larger number of cases the tongue is covered with a thin, dry, light-brown, quite glazed and shiny coating, and presents no feature in common with the so-called typical tongue, except in the freedom from fur of the tip and edges.

The degree of coating of the tongue does not appear to bear any very close relation to the severity of the attack. A heavily furred tongue is often met with in patients who ultimately recover and a lightly furred tongue in the most severe and fatal cases. The brown and glazed tongue more often than not indicates a severe attack; it is apt to pass on into the "typhoid" tongue, which will be referred to later, and this is usually accompanied by a general typhoid condition, from which patients rarely rally. In a few cases in which diarrhœa was a prominent symptom, either during the acute period or in convalescence, the tongue was moist, bright red, glazed, and appeared to be to some extent denuded of epithelium.

One of the most highly characteristic features of the tongue in plague appears to me to be the very marked and rapid changes which it may undergo. A tongue which to-day may be typical may be quite atypical to-morrow, and *vice versa.* A thick coating of fur may almost completely disappear in twenty-four hours, and it may again appear in the course of another twenty-four hours. Sometimes the thick fur on the dorsum becomes brown and glazed in patches. A common occurrence is for the white-gray fur on the dorsum to dry up and become brown on each side, but not in the middle, which remains white or gray; the tongue has in such cases five distinct zones from side to side—viz., pink edge; dry, brown, and caked fur; white or gray centre; dry, brown, and caked fur; pink edge. This change seemed to be a very common and characteristic one. Rather less commonly it is the centre zone which turns dry and brown, leaving lateral zones of gray and white.

When the disease terminates in recovery, the tongue clears slowly or peculiarly rapidly. In the latter event, the fur, with or without a preliminary change to the dry, brown, and caked condition, may peel off in quite large flakes, which leave behind them pink, moist, rather raw-looking patches. This rapid peeling of the tongue in flakes cannot, unfortunately, always be regarded as forerunning recovery. It may occur in the acute period as one of those sudden and apparently inexplicable changes which, as just stated, are characteristic of the disease. It may even precede death by only a few hours. In a severe case which will be quoted later (Case XI. of this paper) the tongue cleared in this way on the seventh day of the disease, with no real improvement in the patient's condition, and the patient died eight days later. In the following case the tongue, which had been "typhoid" in character, was peeling in large flakes at the time death occurred.

CASE V. (Register No. 850.)—Christian, male, aged twenty-three years, admitted March 13, 1898, with a history of eleven hours' illness. The temperature on admission was 105.2°; the tongue was coated with a thick, white fur. There was a left axillary bubo, and six days later a left inguinal bubo appeared. There was some vomiting, which yielded to treatment, on March 16, and on the 17th the tongue was cleaner. On the 20th (a fresh bubo having appeared on the 19th) the tongue was dry, caked, and cracked, and dry sordes had collected around the teeth. On the 22d the pulse was very rapid (164) and weak; there was great emaciation and pallor. There were profuse moist sounds over the lungs. The urine was free from albumin throughout. On the 23d